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The Evaluation of Subclinical Left Ventricular Systolic Dysfunction in Prediabetic Patients with a New Echocardiographic Modality (AFI, Automated Function Imaging) Method

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Objective: Diabetic cardiomyopathy is a major complication of diabetes which has high morbidity and mortality. Diabetic cardiomyopathy is defined as the heart failure resulting from left ventricular systolic and diastolic dysfunction which is independent from factors such as coronary artery disease and hypertension. Prediabetes is known as a strong risk factor for the development of diabetes which needs a long time before formation of diabetes. Early evaluation of cardiac function is important for the prevention of target organ damage in prediabetic individuals. The aim of this study is to evaluate subclinical myocardial dysfunction with a new echocardiographic modalities method, namely speckle tracking echocardiographic method (AFI), in prediabetic patients who has preserved left ventricular systolic function which is evaluated by using conventional echocardiographic method.
Methods: 78 subjects (41 with prediabetes and 37 as control) were included in this study. All subjects' conventional, tissue Doppler and longitudinal strain based on speckle tracking imaging (AFI) data were evaluated by echocardiographic methods.
Results: There were no significant differences between the two groups from the aspects of age, gender, smoking, BMI and blood pressure. The mean longitudinal systolic strain values (19,22% ± 2,68% vs 20,37% ± 2,02, P=,034) and the ratio of Em/Ea (1,03 ± 0,22 vs 1,29 ± 0,44, P=,002) were found significantly lower in prediabetic patients than those in controls, whereas the ratio of E/E'(9,53 ± 2,3 vs 7,61 ± 2,59, P=,001) was significantly higher. The mean cholesterol (220,22 ± 47,23 vs 191,41 ± 38,83, P=,005), LDL (147,06 ± 40,43 vs 119,93 ± 32,76, P=,002) and trigliserid (155,20 ± 107,06 vs 88,11 ± 59,53, P=,001) levels and the measures of mean height (169,00 ± 8,66 vs 165,11 ± 7,38, P=,037), weight (78,07 ± 13,31 vs 71,92 ± 13,77, P=,048) and waist circumference (90,50 ± 9,70 vs 84,70 ± 10,08, P=,012) were statistically higher in prediabetic patients compared with the ones in control group.
Conclusion: The results of this study revealed that the subclinical LV systolic dysfunction may develop also in the period of prediabetes before the development of diabetes. Detection of subclinical LV systolic dysfunction in prediabetic individuals will contribute to determine high-risk groups for the development of diabetes and its complications, and also to specify health policies regarding to take preventive eactions.

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Comparision of Papillary Muscle Deformation Parameters in Patients with Hypertrophic Cardiomyopathy and Hypertension

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Background: The aim of the present study was to analyze anterolateral papillary muscle longitudinal strain (ALPM-St), posteromedial papillary muscle longitudinal strain (PMPM-St), anterolateral papillary muscle time to peak longitudinal strain (ALPM-time), posteromedial papillary muscle time to peak longitudinal strain (PMPM-time) and asynchrony between papillary muscles in patients with hypertrophic cardiomyopathy (HCM), hypertensive left ventricular hypertrophy (H-LVH) and normal subjects using echocardiography with 2-dimensional speckle tracking imaging (2D-STI).
Methods: Conventional echocardiography, tissue Doppler imaging, and 2D-STI were performed in 36 patients with HCM, 10 patients with H-LVH (New York Heart Association functional class ≤II and preserved ejection fraction) and 10 normal subjects. All echocardiographic examinations were performed by iE33 and Q-lab version 8.1 (CMQ, Philips inc). The significance of differences between groups was evaluated by non-parametric analysis (Mann–Whitney test).
Results: We summarized the results in Table 1. In patients with HCM, ALPM St was lower than in patients with H-LVH and normal subjects (p=0.009 and p=0.008, respectively). No difference was found between H-LVH and normal subjects (p=0.9). In assesment of PMPM St there was no difference between H-LVH and HCM (p=0.096) but in patients with HCM, PMPM St was lower than normal subjects (p=0.006). When we compared the papillary muscles time to peak longitudinal strain and papillary muscles asynchrony there was no difference between all groups. Global longitudinal strain was lower in patients with HCM when compared with H-LVH group and normal subjects (p <0.0001 and p <0.0001, respectively) but there was no difference between H-LVH and normal subjects group. HCM patients were analyzed in two subgroups: ALPM St in patients with obstruction was lower than in patients with non-obstructive (p=0.025). And there was asynchrony between papillary muscle in these subgroups (obstrutive vs. nonobstructive. p=0.022).
Conclusion: ALPM St is decreased in patients with HCM and H-LVH. Impairment in ALPM St in patients with HCM is greater than in patients with H-LVH. In obstructive HCM patients ALPM St is lower than non-obstructive group. There is no difference between all groups in papillary muscle asynchrony but in HCM subgroups there is a significant difference. Papillary muscle deformation parameters are different in patients with HCM, H-LVH and in normal subjects.

Table 1

Variable	HCM (n=36)	HT (n=10)	Normal (n=10)	p (HCM vs HT)	p (HCM vs Control)	p (HT vs Control)
Age	46.3±14.6	60.4±8.7	42.2±11.2	0.008*	0.49	0.001*
Male/Female	21/15	7/3	8/2	0.93	0.21	0.33
Septal thickness (mm)	24.7±6	16.3±2,4	11.3±1	0.005*	< 0.0001*	< 0.0001*
Posterior wall thickness (mm)	13.1±2.6	14.5±1.6	10.6±0.52	< 0.0001*	0.002*	< 0.0001*
Septum/posterior ratio	1.94±0.53	1.12±0.09	1.07±0.08	0.076	< 0.0001*	0.28
LV mass (gr)	406.8±111.7	303.2±61.5	181.4±29.8	< 0.0001*	< 0.0001*	< 0.0001*
LV end-diastolic diameter (mm)	44.8±6.3	46.2±5.5	45.5±3	027	0.67	0.32
LV end-systolic diameter (mm)	24.7±5	26.3±5.1	25.4±2.4	0.42	0.53	0.74
Mitral E velocity (cm/s)	0.76±0.18	0.6±0.12	0.7±0.13	0.013*	0.35	0.14
Mitral A velocity (cm/s)	0.61±0.21	0.7±0.11	0.64±0.14	0.07	0.49	0.11
Mitral E Deceleration Time (ms)	193.4±64.3	225±54.4	220±50.7	0.90	0.15	0.80
Mitral Septal Sa (cm/s)	7.3±2.1	6.6±1.01	8.7±1.6	0.31	0.07	0.01*
Mitral Septal Ea (cm/s)	5.06±1.6	5.5±1.7	9.4±3	0.49	< 0.0001*	0.003*
Mitral Septal Aa (cm/s)	7.5±1.8	8±1.04	10.3±1.8	0.53	< 0.0001*	0.007*
Mitral Lateral Sa (cm/s)	8.2±2	7.3±1.1	9.5±1.7	0.19	0.11	0.005*
Mitral Lateral Ea (cm/s)	8.7±3.1	6.8±2.5	11±3.1	0.09	0.06	0.019*
Mitral Lateral Aa (cm/s)	8.8±2.5	9.9±2.7	9.4±1.8	0.26	0.36	0.54
Mitral Septal E/Ea	16.2±6.1	11.9±3.3	8.1±2.8	0.09	< 0.0001*	0.019*
Mitral Lateral E/Ea	9.5±3.5	9.9±2.9	6.8±2.0	0.65	0.009*	0.014*
Global Longitudinal Strain (%)	-10.8±2.7	-14.6±2.3	-16.3±2.0	< 0.0001*	< 0.0001*	0.12
Global Radial Strain (%)	11.9±4.5	18.5±6	18.5±4.8	0.001*	0.001*	0.35
Global Circumferential Strain (%)	-13.9±3.3	-15.1±2.5	-16.2±1.8	0.35	0.001*	0.45
Anterolateral Papillary Muscle Strain (%)	-16.7±5.6	-22±5	-21.7±3.6	0.009*	0.008*	0.91
Anterolateral Papillary Muscle Time to Peak Strain (ms)	45±11	44±12	44±7	0.82	0.74	0.97
Posteromedial Papillary Muscle Strain (%)	-17±5,9	-20.7±6.7	-22.5±6.4	0.10	0.006*	0.85
Posteromedial Papillary Muscle Time to Peak Strain (ms)	45±11	45±11	48±6	0.78	0.50	0.80
Interpapillary Muscle Asynchrony (ms)	9±8	10±9	5±5	0.65	0.17	0.22

Clinical and echocardiographic characteristics of patients (* p <0.05)